SAT0465

CLINICAL MANIFESTATIONS AND PROGNOSIS OF SLE WITH CLINICALLY SIGNIFICANT ANTIPHOSPHOLIPID ANTIBODIES

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Background: Antiphospholipid antibodies (aPLs) have been described in 20% >40% of SLE patients, with 50%>70% of patients with SLE and aPL showing the clinical features of APS after 20 years of follow-up¹. It assumes that aPLs-positive SLE patients would have a more severe clinical phenotype and worse prognosis than those without aPLs.

Objectives: We decided to investigate the clinical manifestations and prognosis of SLE with clinically significant aPLs in a multiple centre SLE cohort.

Methods: A follow-up study to investigate the prognosis of SLE, has been conducted in 26 centres across Jiangsu province as described before². SLE patients fulfilled ACR/SLICC criteria. Patients who had ever recorded first admissions and detected aPLs during the 1999±2009 decade were followed and checked for their survival status in 2015. Clinically significant aPL were defined as: positive LA test, aCL IgG/IgM antibodies>99 th percentile and/or aβ2GPI>99 th percentile on two or more occasions at least 12 weeks apart.

Results: 1) Among 1372 SLE patients, 495 patients was reported the aPLs minutely, and 146 cases was with clinically significant aPLs. Compared with aPLs negative SLE patients, the proportion of men, and the rates of oral ulcer, neuropsychiatric involvement, dsDNA, antinuclear antibody and C3 were significantly higher in aPLs positive SLE. (table 1)

	aPLs positive n=146	aPL negative n=349	P
Gender (male)	5	32	0.017
Oral ulcer	29	44	0.047
Thromboembolism	16	18	0.031
Neuropsychiatric	14	20	0.000
C3 decrease	119	248	0.015
Anaemia	109	214	0.005
ANA positive	138	317	0.048
Anti dsDNA positive	83	169	0.009

2) There were 20 deaths in aPLs positive SLE group and 52 deaths in aPLs negative SLE group during the average follow up of 7.38±0.56 years and 7.54±0.47 years respectively. There was no significant difference in survival curves by Kaplan Meier survival analysis (p=0.776). (Picture 1)

3) Multivariate Cox regression analysis revealed that long time of diagnosis (HR 4.205, p<0.05), SDI>1 in admission (HR 11.982, p<0.01), neuropsychiatric involvement (HR 2.826, p<0.05), and increased serum creatinine (HR 8.403, p<0.01) were independent predictors of mortality. (table 2)

factors	Univariate			Multivariate		
	HR	95% CI	P	HR	95% CI	P
Time of diagnosis>1 year	5.26	2.24–12.32	0	4.20	1.06–16.56	0.04
SDI≥1 on admission	4.37	1.67–11.40	0.003	11.98	2.85–50.25	0.001
Neuropsychiatric	4.52	1.89–10.84	0.001	2.82	1.01–7.85	0.048
Increased serum creatinine	4.39	1.87–10.30	0.001	8.40	2.20–32.04	0.002

Conclusions: In this study, we observed that around one-third of patients had clinically significant aPLs, and such autoantibody positivity was associated with a different clinical and serological profile. However, the mortality between aPLs

positive and negative SLE patients had no significant difference. SLE patients presented with vital organ damages rather than active disease at initial hospitalisation are likely to have a poor outcome, especially neuropsychiatric involvements and renal insufficiency.

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SERUM VITAMIN D DEFICIENCY IS ASSOCIATED WITH ACTIVE RENAL DISEASE IN SYSTEMIC LUPUS ERYTHEMATOSUS

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Objectives: 25-hydroxyvitamin D (25(OH)D) deficiency is common in systemic lupus erythematosus (SLE) as well as chronic kidney disease. In this study, we investigated the association of 25(OH)D deficiency and renal involvement in SLE patients.

Methods: Two hundred seventy-two SLE patients and 138 control subjects were enrolled; 102 patients with active nephritis, 42 patients with inactive nephritis, and 128 patients with non-renal disease. Serum 25(OH)D levels were measured, and clinical and laboratory data were obtained from medical records.

Results: Mean serum 25(OH)D levels were significantly lower in SLE patient than control subjects (19.6 ng/ml versus 21.7 ng/ml, p=0.006). Out of 272 patients, 61.8% were vitamin D deficient (defined as <20 ng/ml). Patients with active nephritis had lower serum 25(OH)D levels (16.9 ng/mL) than patients with inactive nephritis (20.2 ng/mL) and without nephritis (21.5 ng/mL) (p=0.030, p<0.001), but there was no difference between the inactive nephritis and non-renal disease. Moreover, serum 25(OH)D levels were positively correlated with complement C3 (γ=0.135, p=0.026) and C4 (γ=0.159, p=0.009), but inversely with anti-dsDNA antibody level (γ=-0.156, p=0.010). Analysis of receiver operating characteristic curve for differentiating active nephritis and non-renal disease revealed an area under the curve (AUC) of 0.676, which is better than those of anti-dsDNA antibody (AUC=0.585, p=0.038) and complement C3 (AUC=0.509, p=0.001), C4 (AUC=0.538, p=0.008).

Conclusions: Vitamin D deficiency is more common in SLE patients with active nephritis, and its level could be a potential marker for active renal disease in SLE. A prospective cohort study is needed to further elucidate the causal relationships over time.

Disclosure of Interest: None declared

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SAT0467

CEREBRAL VENOUS THROMBOSIS OCCURRENCE IN SYSTEMIC LUPUS ERYTHEMATOSUS WITHOUT ANTIPHOSPHOLIPID ANTIBODY SYNDROME: A MONOCENTRIC SERIE OF 10 CASES

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Background: Cerebral venous thrombosis (CVT), which includes cerebral vein and dural sinus thrombosis, is a rare disorder that can lead to significant morbidity and mortality. Its occurrence in SLE in the absence of APS has been rarely reported. In this study we aimed to describe a cohort of SLE patients suffering from CVT without APS.

Methods: We collected retrospectively clinical and biological data of patients with confirmed CVT in the Pitié-Salpêtrière cohort of SLE (n=1352 patients). Patients fulfilled ACR SLE criteria. The diagnosis of CVT was confirmed by brain imaging studies. Exclusion criteria were patient with a lupus anticoagulant or IgG/IgM anti-cardiolipin antibodies or anti-β2 glycoprotein-1 abs. We searched on PUBMED database for case report of this association published in English until 31 August 2017. Lupus flares were defined according the SELENA Flare instrument.

Results: We included 10 patients (8 women and 2 men). The median (range) age at diagnosis of CVT was 28 years.⁹⁻⁵³ The CVT occurred with a median delay of 4